Antiphospholipid antibodies: crossroads between autoimmunity and infections?

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We read with interest the letter by Ponzetto and co-workers [1] commenting on our recently published Point of View paper [2]. They propose the hypothesis that testing for Helicobacter pylori (HP) and, when positive, managing that underling infection might improve the current management of some patients with antiphospholipid syndrome (APS). Of note, they highlighted that HP's eradication might result in loss of antiphospholipid antibodies (aPL)[3]. There are several mechanisms through which pathogens can initiate or perpetuate autoimmunity and, while the factors causing production of aPL remain undefined, there is evidence that molecular mimicry is one of the mechanisms by which experimental APS can occur in association with certain pathogens [4]. However, when reviewing the available data on the topic, Abdel-Wahab at al. showed that, although the development of aPL with all traditional manifestations of APS were observed after variety of infections (mainly chronic viral infections including HIV and HCV), the causal relationship between infections and APS is still on debate [5]. Clinical evidences support that infections can trigger the development of catastrophic APS [6]; nevertheless, the possible contribution of various infections in the pathogenesis of APS need further longitudinal and controlled studies to establish the incidence, and to better quantify the risk and the outcomes of aPL-related events after infection.

Finally, an important question still remains: is the aPL negativizationenough to change the therapeutic approach in a patient with APS? Recent data suggest that persistent negative aPL profile is not an indication to interrupt oral anticoagulant therapy as patients are still at high risk of recurrences [7].

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